CLINICAL LECTURES ON CIRCULATORY AFFECTIONS.—LECTURE II. THE CAUSE OF THE PRESYSTOLIC MURMUR.

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CLINICAL LECTURES ON CIRCULATORY AFFECTIONS. —LECTURE II. THE CAUSE OF THE PRESYSTOLIC MURMUR.

By G. A. Gibson, M.D., D.Sc., *Physician to the Royal Infirmary, Edinburgh.*

Since the discovery of the purring thrill by Corvisart,[1] and of the presystolic murmur—which Bertin[2] previously heard but misunderstood—by Fauvel,[3] the explanation of the murmurs of mitral obstruction has been the subject of much discussion. The greatest differences of opinion as regards the clinical features of this valvular affection are centred in the presystolic murmur, and it is to this subject that your attention will be specially directed to-day.

You have been taught that there is considerable variability in the results of auscultation in mitral obstruction, and you have, as might be expected amongst so many female patients, had ample opportunities of becoming conversant with the different murmurs which occur. You are therefore familiar with the facts that in mitral obstruction there may be an apical murmur immediately following the second sound—usually termed diastolic; or occurring a little later—therefore sometimes called post-diastolic; or taking place immediately in advance of the first sound—the well-known presystolic murmur. As regards the existence and character of these murmurs, all observers are at one, but when we approach their explanation we find divergent views, at least as regards the presystolic murmur.

The usual explanation of these different murmurs is that those of diastolic and post-diastolic rhythm are produced by the active diastole of the left ventricle drawing blood from the

[1] "Les maladies et les lesions du cœur," Paris, 1806, p. 236.
[2] "Maladies du cœur," Paris, 1824, pp. 176 and 186.
[3] *Arch. gén. de méd.*, Paris, 1843, Sér. 4, tome i. p. 1.

uricle, but aided, no doubt, by the general onward current the blood, impelled through the pulmonary artery and ulmonary veins by the right ventricle, which in such cases is ways hypertrophied. The presystolic murmur has been, and , generally considered as due to the contraction of the left uricle, and its frequent absence, or disappearance and reappearnce, attributed to varying degrees of weakness of this part of the eart. This current explanation of the presytolic murmur only ates from the observations of Gairdner,[1] who accurately defined s relations to the different events of the cardiac cycle. Gairdner's iews were strongly supported and his observations amplified a w years later by Fagge[2] and Balfour,[3] and the great majority authors who have dealt with the subject have espoused their iews, although certain of them, to be more particularly discussed ter, have proposed certain modifications of the explanation.

The first expression of doubt regarding Gairdner's explanation me from Ormerod, who in the Address on Medicine at the Camridge Meeting of the British Medical Association, in 1864, uttered e following remarks[4]:—

"Thus, to take a common instance: a murmur audible at or ear the apex only, accompanying the first sound, rising in tensity with the continuance of the first sound, and ending uddenly with a snap at its closure, admits of more than one nterpretation. Its seat is unquestioned—the mitral or the icuspid orifice. But is it due to obstruction to the flow of lood from the auricle to the ventricle during the auricular ystole, or to imperfection of the auriculo-ventricular valve, llowing regurgitation during the contraction of the ventricle? o the first explanation we may object that a direct mitral murmur, such as is generally admitted to be of this nature, is uite unlike the murmur under consideration. It is soft, and losely resembles the murmur of sigmoid regurgitation. Such a murmur is very rare, while the murmur under consideration is ot at all uncommon. It may be questioned, too, whether the ontraction of the auricle, slight and momentary as experimentalists report it to be, is capable of producing so loud a ound. To the second explanation, I do not see that there is ny insuperable objection. It assumes that the coaptation of ll the portions of the valves is not simultaneous; but that vhen, at a certain point of contraction, the valve is closed, the egurgitant stream is cut off, and the cessation of the murmur coincides with the sudden jerk of valvular tension. In favour of his view, it may further be urged that this peculiar murmur is

[1] "Clinical Medicine," Edin., 1862, p. 574.
[2] *Guy's Hosp. Rep.*, Lond., 1871, vol. xvi. p. 247.
[3] "Clinical Lectures on Diseases of the Heart and of the Aorta," London, 1876, . 101.
[4] *Med. Times and Gaz.*, London, 1864, vol. ii. p. 153.

not always a sign of organic disease. It is often a murmur of palpitation, where such disturbance of the rhythm of the heart's contraction is singularly liable to occur." Ormerod, in addition to being somewhat indefinite in these statements, and absolutely wrong in the final remark quoted, is far from sure of his own views, for a little further on, speaking of the two interpretations, he states: "I feel that it is scarcely possible to determine absolutely which is the correct one, whether the murmur is direct or regurgitant."

Barclay[1] and Charlwood Turner[2] espoused the opinion of Ormerod. The former in his eloquent discussion of the matter based his arguments largely upon anatomical and physiological evidence. He pointed out that the ventricles begin their contraction before the commencement of the first sound, and that part of the ventricular contraction must therefore be apparently presystolic. He further stated that a double murmur at the apex is very rare, *on account of the diastole being purely passive.* He mentions also that he could scarcely conceive the mitral valve to be so altered as to cause audible vibrations in the onward current while it remained competent. He, lastly, refers to the absence of valves in the pulmonary veins as a stumbling-block to the theory of Gairdner. It is impossible not to admire the modesty of Barclay in bringing forward his views, for he only suggests his own honest conviction that the current explanation was inadequate and that the new view was deserving of consideration. In the same volume Barclay's views were subjected to a thorough criticism by Balfour.[3] Turner in his ingenious reasoning entered very fully into the crescendo character of the murmur, and the close blending which it shows with the subsequent first sound.

Somewhat later, Dickinson[4] came to the support of these authors, and based his advocacy of the ventricular causation of the murmur upon the fact that if while listening to it a finger be pressed between the ribs, "the beginning of the murmur will be found synchronous with the beginning of the impulse." Dickinson in illustration of his view, employed a light pin with a big head inserted in a cork fixed to the chest wall at the position of the apex-beat, whose movements were watched while auscultating with a binaural stethoscope. Believing that the movements of the pin were systolic, he concluded: "By this showing the murmur does not precede but begins with the systolic movement, and is not presystolic but systolic." He allowed, nevertheless, very candidly the thrill and absence of scapular conduction of the murmur to be in favour of the orthodox view. In conclusion, he emphasises three points in support of his view—(1) The intensity of the

[1] *Lancet*, London, 1872, vol. i. pp. 283, 353, 394.
[2] *St. Thomas' Hosp. Rep.*, London, 1876, vol. vii. p. 199.
[3] *Lancet*, London, 1872, vol. i. p. 714.
[4] *Ibid.*, 1887, vol. ii. pp. 650, 695.

murmur; (2) its relation to the carotid pulse; and (3) the presence of a diastolic, or truly obstructive, murmur in many cases along with the presystolic murmur. These views have been supported by M'Vail,[1] and also by Tripier and Devic.[2] They are also held by an important recent author, but as he has advanced certain modifications in the explanation, his views will be considered later.

As has already been remarked, the orthodox view, if it may so be termed, has been subjected to some modification by certain authors, two of whom, Cowan[3] and Gibbes,[4] must be discussed. Cowan, in a paper of high scientific value, believes it to be impossible that the murmur can be regurgitant, and he considers it to be mainly ventricular diastolic in rhythm, but also partially ventricular systolic. He states that the apical impulse cannot be considered as exactly synchronous with the commencement of ventricular systole, as a certain period must elapse before the contraction can cause the impulse to be felt on the chest wall; but as the impulse is felt at the same moment that the first sound is heard, the ventricular systole must have commenced before the first sound is audible or the apex impulse felt. He considers, therefore, that during the period when this murmur occurs, two phenomena are taking place—during the earlier portion the auricular systole, and during the terminal part the beginning of the ventricular systole. He is of opinion that the auricular systole must play an important part in causing this murmur, although various writers have doubted this, on the assumption that the slender auricle can exert but little force, but he recalls the facts that the systole begins in the pulmonary veins, and that when the pressure in the ventricle is nil or negative, there is a positive pressure in the pulmonary arterioles, capillaries, and veins, arising from right ventricular systole. He therefore concludes: "Thus the murmur of mitral stenosis is caused in its earlier portion by the blood driven through the thickened valves, by the auricular systole, and in its terminal part by blood flowing into the ventricle, whilst changes in the tension of the valves and alterations in the lumen of the orifice are taking place, the result of the contraction of the ventricle as a whole, and of the musculi papillares in particular." The other author, Gibbes, in some very interesting contributions, considers that the presystolic murmur is composed of three parts — audible right ventricular vibration; the auriculo-systolic murmur; and the slapping first sound. He holds that in mitral obstruction the two ventricles do not act simultaneously, and that part of the

[1] *Brit. Med. Journ.*, London, 1887, vol. ii. p. 786.
[2] "Traité de pathologie générale (Bouchard), Paris, 1897, tome iv. p. 257.
[3] *Glasgow Med. Journ.*, 1898, March.
[4] *Clin. Journ.*, 1899, July 12: and *Med. Times and Hosp. Gaz.*, London, March 10, 1900.

systole of the right ventricle occurs while the left ventricle is in diastole. These muscle vibrations are regarded by Gibbes as the cause of the crescendo character of the murmur and its blending with the first sound. The portion of the murmur produced by contraction of the auricle he considers to be often absent in consequence of feebleness of the muscle, and he appears to think this is one of the later events in the evolution of the obstruction requiring the orifice to be considerably contracted for its production. The slapping first sound is considered by him as an integral portion of the phenomena, and he considers the muscle vibrations as the cause of the peculiar and intimate connection between the murmur and the sound.

Brockbank[1] has ranged himself upon the side of the heretical theory, as he terms it, and in several most interesting papers he has very fully given the reasons for separating himself from those who believe in orthodox views. His conception is that "the crescendo murmur of mitral stenosis is produced by blood regurgitating through the stiff, rigid orifice of the narrowed valve, while this orifice, which resists closure, is being rapidly diminished in area and finally obliterated by the action of a strong force, which on its part increases rapidly in strength with the duration of the bruit." By the most ingenious mechanism he has succeeded in imitating the presystolic murmur, and with this apparatus he has found it quite easy to mimic the "blubbering" character of the presystolic murmur while fluid was forced, as it were, from ventricle to auricle, while, with the current reversed, it was impossible to produce any imitation of it. Brockbank is of opinion that any theory which explains the production of a crescendo murmur must hold good, no matter what the condition of the left auricle may be, and he states that the murmur not uncommonly occurs in cases in which the auricle is dilated and weak.

We have therefore to consider the original theory propounded by Gairdner, and its modifications by Cowan and Gibbes, as well as the opposite theory of Ormerod, Barclay, and Dickinson, together with its amplifications by Brockbank.

The chief difficulty which seems to have prevented the universal acceptance of Gairdner's views lies in the conception that the left auricle is too feeble to give rise to anything like the ordinary presystolic murmur. The presystolic murmur is always most distinctly developed, at least in my own experience, in comparatively early cases of mitral obstruction, and particularly in those which are found at a later stage to have the obstruction produced by vegetations on the cusps, without much narrowing of the orifice. The opinion has been forced upon me by the result of much pathological observation, that in the typical button-hole obstructive mitral orifice the murmur is much more commonly

[1] *Med. Chron.*, Manchester, 1897, vol. vii. p. 161; *Edin. Med. Journ.*, 189 vol. v. pp. 236, 341; "The Murmurs of Mitral Disease," Edin. and London, 189

long diastolic one, dying away distinctly before the first sound. We must not shrink from facing the difficulty as to the true presystolic murmur, and we must admit that even when the left auricle is hypertrophied to its extreme possibilities, it is but a feeble organ. Nevertheless it must be remembered that on inspection of the heart after death, the state of the auricle does not, in most cases, bear any necessary relation to its condition at an earlier period. In the cardiac failure leading to the fatal result, the thin auricles undergo a greater degree of dilatation and attenuation than the rest of the heart. In cases with a small mitral orifice, the aspiratory action of the left ventricle is amply sufficient to produce the diastolic murmur, and when the orifice is much constricted, it is probable that the filling of the ventricle will be considerably delayed, so that this diastolic murmur may be prolonged into the period of auricular systole.

While greatly admiring the ingenuity of those who have ranged themselves in opposition to Gairdner's theory, it has not been possible for me to accept their views. The one simple fact that the presystolic murmur may begin as a diastolic murmur with the second sound, and be continued, *without appreciable alteration in its character*, up to the first sound, to my mind entirely negatives the regurgitant theory of the presystolic portion, which would require an absolute revolution in our knowledge of the mechanism of the heart.

The fact that an impulse may sometimes be felt along with the presystolic murmur seems to have influenced the views of Dickinson, and has led him to the belief that the impulse was necessarily that of ventricular systole. Although by no means a warm believer in the utility of the cardiograph for practical purposes, it nevertheless must be stated that in an ordinary cardiogram the systole of the auricle makes itself clearly seen even in cases in which it is not hypertrophied; and if such be one of the chief physical arguments upon which the majority of those who hold that the murmur is regurgitant base their views, it can hardly be regarded as affording good ground for the theory.

The classical case of tricuspid obstruction published by Gairdner seems to be absolutely destructive of the regurgitant theory; but Brockbank, in his criticism of the case, claims that, with the tumour obstructing the orifice, a murmur of regurgitation would result, owing to blood escaping through the gaps in the imperfectly closed curtains, or between the curtains and the tumour. As the obstruction was being gradually forced out from between the valve curtains, the extent of the incompetency of the latter would diminish until complete competency were regained. Considering the whole mechanism of the auriculo-ventricular cusps, it is not possible to accept this view.

As regards the ingenious modification of the orthodox explana-

tion of the murmur, which has been advanced by Gibbes, it seems to me that some of the same objections must be raised to part of his views as have been advanced in regard to the regurgitant theory of the murmur. Gibbes believes that the two ventricles do not act synchronously, and that the right ventricular systole occurs while the left is still in diastole. He attributes part of the presystolic murmur to audible muscle vibrations produced by the contraction of the right ventricle. Now, it seems to me that in those cases which present a diastolic murmur, passing by insensible gradations into a presystolic murmur, we cannot assume that the phenomena begin with the aspiratory force of the left ventricle and end with an early contraction of the right ventricle. There can be no doubt that the right ventricle in many of these cases is greatly hypertrophied, but it has yet to be proved that the various upholders of the theory of hemisystole are correct in their views.

The interesting modification of the orthodox view which has been advanced by Cowan, seems to me undoubtedly worthy of acceptance. The presystolic murmur runs absolutely into the succeeding first sound, and there can be no doubt that changes in the position of the cusps and modifications in the form of the orifice occur, thus leading to the characteristic conclusion of the presystolic murmur. Unfortunately Cowan's views did not see the light until the portion of my work on the heart dealing with this subject had passed through the press, and it was therefore impossible for me, in it, to criticise his opinions; but it is a pleasure now to be able to state my belief that his contribution has added materially to our comprehension of this difficult subject.

As an illustration of the subject now occupying our attention, let me shortly bring before you the facts regarding one of the patients now in our wards. Her case is of much interest in many ways, but chiefly on account of the opportunity which we now have of comparing her present condition with that observed when she was under my care in our wards three years ago.

The patient, æt. 33, and married for thirteen years, has had six children and one miscarriage. She was admitted to Ward 27 on April 21, 1897, complaining of palpitation, breathlessness, and cough, from which she had suffered for three years. She had been much worse since the miscarriage, which occurred six weeks before. She had whooping cough, measles, and smallpox as a child. At the age of 16 she had chorea, and was in the Royal Infirmary for six weeks. She perfectly recovered from this, had no heart symptoms, and remained quite strong till three years ago, when her cough began. The cough had persisted ever since, but had not been severe till recently. Her father died ten years before, æt. 49, of Bright's disease. Her mother was alive and well, æt. 60. Four brothers were dead, one alive. Three died in infancy, and one of enteric fever when æt. 21. She had three sisters alive and strong. Her illness began six weeks before, after the miscarriage. She stayed in bed two weeks after it, then got up and went about for a

week, but the cough became so troublesome that she had to go back to bed. She was treated at home from the Cowgate Dispensary, but, not improving, was admitted to our ward. The patient was thin, pale, and marked by smallpox. Her weight was 7 st. 7½ lb., and her height 5 ft. 3 in. The temperature was never above 99°, and was usually below normal.

The *Alimentary system* calls for no remark, as, although the appetite was very poor, there were no evidences of disturbance.

The *Circulatory system* showed some subjective sensations, such as palpitation and dyspnœa on exertion. Five weeks before, the patient fainted on two occasions. She used to have pain about the præcordia, passing up to the left shoulder, and tightness about the chest.

Pulsation was evident in the fifth interspace, 3½ in. from the mid-sternal line. Pulsation was also seen in the epigastrium, synchronous with the apex-beat.

Palpation confirmed inspection, and revealed a presystolic thrill at the apex. Pulsation was also felt slightly in the second, third, and fourth interspaces.

The upper border of the heart was found at the level of the third rib; the left border was 4½ in. from the midsternal line in the fourth interspace, and the right border was 1½ in. to the right of the mid-sternum.

In the mitral area there was a long, rough, continuous diastolic and presystolic murmur, leading up to an explosive first sound, with a reduplicated second sound. In the tricuspid there was a sharp first and a reduplicated second sound. In the aortic and pulmonary the second sound was reduplicated, and the pulmonary element was accentuated.

The pulse averaged 80 per minute, and was regular in time and force, with low pressure and healthy vessels.

Respiratory system.—The chest wall was thin, and there was more hollowing above and below the right clavicle than the left. The respiration was of costo-abdominal type, with a rate of 22 per minute. The movements at the right apex were deficient. Vocal fremitus was increased over the right apex. There was impaired percussion sound over the right apex. Posteriorly there was some impairment of the sound down the whole of the right side. Expiration was prolonged at the right apex. Vocal resonance was everywhere increased, and whispering pectoriloquy was marked at the right apex. A few sibilant rhonchi were heard over both lungs, especially with inspiration.

Urinary system.—There was a trace of albumin in the urine.

The treatment consisted in absolute rest. Strophanthus and strychnine were administered, also iron and arsenic, and the patient improved very much in every way.

She left the Infirmary on May 3rd on account of home troubles. She felt much better, but the pulse was still feeble, and she looked very anæmic. Her weight was 8 st. when she left.

The patient was re-admitted to Ward 27 on June 13, 1900, complaining greatly of breathlessness and of a pain passing across her heart "like the shot of a gun."

Circulatory system.—The cardiac impulse was very diffuse, and there

was marked epigastric pulsation. The diffuse cardiac impulse had on palpation a heaving character, and was preceded by a slight apical thrill. The apex-beat was in the fifth interspace beyond the mammillary line. The upper border of the heart was at the upper border of the third rib; the left border was 5½ in. to the left of the midsternum; and the right border was 1½ in. to the right of it.

In the mitral area, the first sound was obscured by a harsh blowing murmur. The second sound was only feebly heard, and seemed to be reduplicated. It was followed by a distinct diastolic murmur. In the tricuspid area, the first sound was closed, and the second reduplicated. The first sound in the aortic area was quite pure; the second was reduplicated. The first sound in the pulmonary area was rather faint, and the second markedly accentuated and reduplicated.

The pulse was 96; regular in time and force; very compressible and small. The vessel wall was healthy.

Respiratory system.—The respirations were 28 per minute; regular, but somewhat shallow and laboured. There was a great deal of cough, with considerable quantities of sputum. This was dark red in colour, somewhat frothy and viscid, not altogether unlike pneumonic sputum. Neither pneumococci nor tubercle bacilli were present. The chest wall was thin. The movements of respiration on the right side were lessened. The percussion sound was impaired at the right apex. Posteriorly, there was an area of dulness about 3 in. square immediately below the angle of the scapula on the right side. Elsewhere the sound was fairly good. Anteriorly, on both sides, there were some sibilant râles and rhonchi to be heard both on inspiration and expiration, accompanying the breathing, which was harsh vesicular in type. Over the dull area on the right side posteriorly the breathing was markedly tubular, with increased vocal resonance.

Urinary system.—The urine was scanty and high-coloured. It had a deposit of urates, but no other abnormal constituents.

The hæmoptysis stopped on June 27th, and the sputum became a white viscid mucous substance.

The patient, although still very far from well, insisted on leaving hospital on July 2nd, on her own responsibility. Permission could not be given for her leaving, as she was still very weak, and her general condition such as made it a risk to move her.

You find a considerable contrast between the results of physical examination ascertained three years ago and at the present time. When under my care during her first residence in hospital, there was simply a long rough murmur beginning soon after the second sound, and terminating in a loud explosive first sound, the second sound being doubled. The results of auscultation during her present residence have been entirely different: there has been a short, rough, diastolic murmur and a harsh first sound, followed by a rough, blowing, systolic murmur, the second sound still being doubled.

It is very obvious why this complete change in the result of auscultation has taken place. The mitral valve has become incompetent, either from dilatation of the orifice, increasing

deformity of the cusps, or lessened adaptability of the valvular apparatus. By some such process the regurgitant murmur is allowed. On the other hand, the disappearance of the presystolic murmur is due to the weakening of the left auricle, which leaves the flow of the blood into the ventricle to be mainly sustained by its own aspiratory force, as well as by the contraction of the right ventricle.

It is unnecessary on the present occasion to refer in detail to the physical signs connected with the respiratory system. They may be shortly summed up by stating that the various phenomena are due to venous stasis and pulmonary hæmorrhage.

Since this Lecture was delivered, Gibbes[1] has published another very able contribution upon this subject, characterised particularly by its masterly analysis of the principles and results of physical examination in mitral obstruction, and more especially as regards cardiographic investigation. His observations on the action of the right ventricle in the condition amply reward perusal.

[1] *Edin. Med. Journ.*, 1900, New Series, vol. viii. p. 129.